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# Emergency Management of Chronic Wounds

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As America's emergency departments witness an escalation in care provided to an aging population, the emergency physician increasingly evaluates and treats manifestations of chronic disease. In 2004 the population of Americans over the age of 65 was 34 million. By the year 2030 this population is projected to reach 69 million. Nonhealing wounds are often a presenting manifestation of chronic disease. They are a source of pain and disability for this population. Emergency physicians should possess a fundamental knowledge in the management of chronic wounds. This article familiarizes the emergency physician with the epidemiology of chronic wounds, the physiology of tissue repair, the pathophysiology involved in wound healing failure, the common types of chronic wounds, and specific management strategies.

## Epidemiology of chronic wounds

Acute wounds are wounds that proceed through an orderly and timely reparative process and result in a sustained restoration of anatomic and

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functional integrity. In chronic wounds, however, this process has been disrupted. Chronic wounds fail to proceed through the usual stepwise fashion and experience prolonged or incomplete healing, with lack of restoration of integrity. A chronic wound is generally defined as a wound that fails to progress over a period of 30 days [1]. Some wounds never heal. The financial impact for their treatment along with the emotional and physical impact can be devastating for patients and create a burden on the health care system as a whole. These wounds predominantly affect patients over the age of 60 [1]. It is estimated that chronic wounds affect 0.78% of the population, with a prevalence ranging from 0.18% to 0.32% [2]. The most common forms of chronic wounds are related to diabetes, venous stasis disease, peripheral vascular diseases, and pressure ulcerations. In 1999 it was estimated that the average cost for 2 years of treatment of a diabetic ulcer was $27,987 per patient [3]. More recently the cost for a single ulcer has increased to $8000, with the cost of an infected ulcer increasing to approximately $17,000 per year and the cost of a major amputation spiraling upward to approximately $45,000. In the year 1996 more than 86,000 lower extremity amputations were performed in the United States. Roughly half of these patients develop an infection or ulceration on the contralateral limb within 18 months [4]. The incidence of diabetes is on the rise in the United States. America currently spends an estimated $1.5 billion annually in the treatment of diabetic foot ulcers.

## Normal tissue repair

The process of normal tissue repair is a complex but organized sequence of events mediated by cellular and biochemical activities. This process can be broken down into four characteristic stages. Each stage of repair involves a propagating series of events that lead to the next stage.

### *Stage I. Hemostasis*

The process of hemostasis is initiated with tissue injury. There are three major steps that characterize this process. The first step is the formation of the fibrin clot. On exposure to type I collagen, the inactive proteases of the intrinsic and extrinsic pathways become activated. These activated proteases induce the conversion of prothrombin to thrombin, which in turn induces the conversion of fibrinogen to fibrin. The fibrin strands assemble into a network that traps red blood cells and platelets, forming a clot in the wound that blocks flow from injured blood vessels. Vasoconstriction further reduces the flow of blood from injured vessels. Platelet degranulation occurs in the second step. As the platelets become entrapped in the fibrin lattice, degranulation allows for the release of growth factors. Chief among these are platelet derived growth factor (PDGF), transforming growth factors (TGF), basic fibroblast growth factor (bFGF), and vascular endothelial

growth factor (VEGF). These growth factors are chemotactic to fibroblasts, neutrophils, and monocytes. The third step in hemostasis is the formation of the provisional wound matrix. Type III collagen is synthesized by fibroblasts that serve to reinforce the matrix. More than just a fibrin clot, the provisional wound matrix is a dynamic environment of biochemical signaling. The transition to the next stage occurs as the cells of immune function and tissue repair migrate to the provisional wound matrix.

*Stage II. Inflammation*

The inflammatory phase begins approximately 24 hours after injury and normally lasts up to 2 weeks [5]. With the provisional wound matrix in place, white blood cells migrate to the region in response to chemotaxis. The marginating pool of neutrophils is the first line of defense against infection. Through phagocytosis they ingest and kill bacteria. They also play a role in removing foreign materials and devitalized tissue. Mast cells release histamine, which causes the rubor and calor noted about the periwound tissues. Edema ensues. Various chemokines are released that mediate the recruitment and activation of more neutrophils, eosinophils, lymphocytes, basophils, and macrophages. The T and B lymphocytes exert humoral and cell-mediated immune responses. Following migration into the wound matrix, neutrophils perform several activities. Oxygen free radicals are released that kill phagocytized bacteria. Proinflammatory cytokines are released that perpetuate the overall inflammatory process. Neutrophils also release high levels of proteases known as the matrix metalloproteases (MMPs). These proteases come in the form of elastase and collagenase that remove components of the damaged extracellular matrix within the wound. Activated macrophages play a similar role as the neutrophils early in the inflammatory stage. They ingest bacteria through phagocytosis and secrete MMPs. Late in the inflammatory stage the macrophage plays a key role in mediating the transition to the next stage of repair. By releasing protease inhibitors, macrophages turn off the furnace of proteolytic destruction. Growth factors (PDGF and TGFβ) and anti-inflammatory cytokines are released. A second call is made to recruit and activate fibroblasts to the wound matrix.

*Stage III. Proliferation*

Within the fibrin-rich wound matrix, the fibroblasts proliferate and begin synthesizing collagen, fibronectin, and proteoglycans. With the release of TGFβ and VEGF angiogenesis is stimulated. This combination of activities leads to the development of granulation tissue. Granulation tissue is composed of a density of collagen and capillaries. It serves as a transitional replacement for normal dermis. The process of epithelialization follows in this timed sequence of events.

*Stage IV. Maturation*

The final phase of healing takes place over the next year. Type III collagen is replaced with the deposition of type I collagen. The organization and cross-linking of collagen fibers increases their tensile strength. The density of the capillaries is reduced as the tissues begin to take on the characteristics of normal dermis.

## Pathophysiology of chronic wounds

Unlike the organized sequence of events that occurs during normal tissue repair, the chronic wound environment is suspended in a stage of prolonged inflammation. The studies of wound fluid analysis have led to the understanding of three key concepts with regard to the molecular and cellular activities of chronic wounds. First, chronic wounds have decreased mitogenic activity within fibroblasts, keratinocytes, and vascular endothelial cells [4]. This decrease in cellular activity is known as cellular senescence. Second, chronic wounds have elevated levels of proinflammatory cytokines that perpetuate the inflammatory stage. Finally, chronic wounds have elevated levels of destructive protease activity.

## Common pathways in the development of chronic wounds

Normal wound healing, as outlined previously, may be negatively impacted by numerous factors creating a state of perpetual imbalance. The vast majority of chronic wounds are a consequence of three basic conditions: vascular disease (venous disease, arterial insufficiency), diabetes mellitus, or inappropriate pressures (pressure ulcers). Factors that negatively affect wound healing are listed in Box 1. Inappropriate recognition or

**Box 1. Factors that delay wound healing**

- Advanced age/immobilization.
- Infection, poor hygiene.
- Malnutrition/chronic illness
- Diabetes
- Peripheral vascular disease
- Medications (corticosteroids, immunosuppressants etc.)
- Cigarette smoking
- Stress (mechanical/emotional)
- Inadequate/inappropriate wound care
- Excessive dryness/moisture
- Edema

treatment of the chronic wound may allow the wound to advance to deeper tissue layers, including muscle and bone, and eventually threaten limb and life.

## Examples of chronic wounds

### *Diabetic foot ulcers*

Foot ulcers develop in 15% to 20% of the 16 million people in the United States who have diabetes [6–8] and 85% of lower extremity amputations are preceded by foot ulcers. At least 50% of amputations within the population of people who have diabetes can be prevented [9]. People who have diabetes are prone to arterial insufficiency and generally diminished immunity but neuropathy (which develops in 42% of people who have diabetes after 20 years) is the most critical element in the development of nonhealing ulcers [10–12].

Although not all foot deformities are a direct result of diabetes (eg, bunion propensity is likely genetic), diabetic neuropathy affects motor innervation to muscle spindles and may lead to intrinsic foot muscle weakness [10]. The resulting imbalance between normal extrinsic and weak intrinsic muscle groups may lead to the development of claw or hammer toes, which shift pressure and frictional forces to vulnerable areas, particularly the plantar metatarsal heads. The top (dorsal surface) of the deformity is also at risk from rubbing on the shoe. Sensory and autonomic neuropathic changes give rise to dry, insensate feet unable to protect themselves from low-grade repetitive trauma. Charcot arthropathy may present initially as a diffusely erythematous, hot foot that eventually degenerates into a chronic deformity termed the Charcot ''rocker bottom'' foot. These numb, highly deformed feet are at particularly high risk for ulceration. Acute Charcot is a difficult diagnosis to make but vital to suspect early.

The combination of diabetic neuropathy, foot deformity, and minor trauma has been implicated as a basic recipe for foot ulceration [13,14]. A callus can increase underlying tissue pressure by as much as 30% [15] and may be the first indication of imminent woe [16]. A callus is classified by some practitioners as a pre-ulcerative state [17]. When whitish, macerated tissue appears beneath the callus, ulceration is looming. Diabetic ulcers may be avoidable to some extent through regular foot examination, correction of foot deformities, detection of sensory neuropathy with a monofilament, and identification of sources of trauma, often related to ill-fitting footwear (Fig. 1).

### *Venous stasis ulcers*

Some 85% of chronic lower extremity skin ulcers are related to chronic venous insufficiency (CVI) [18]. It is estimated that 2.5 million Americans

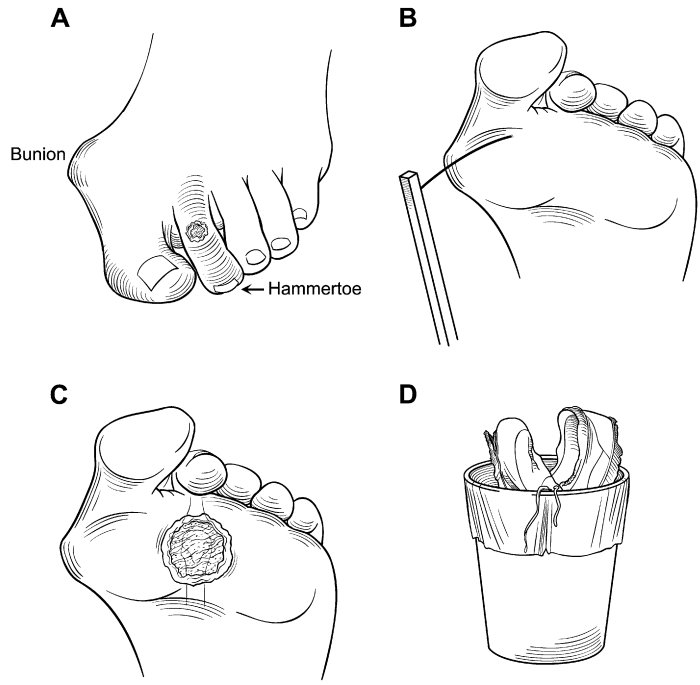


Fig. 1. Diabetic foot deformities allow shoes to rub on top of toe (*A*) and transfer pressure to plantar surface of metatarsal heads (*C*) resulting in callus development and ulceration. Semmes-Weinstein mono-filament (*B*) tests for neuropathy. Ill-fitting shoes (*D*) are a major factor in diabetic ulceration and should be discarded.

have CVI, of whom 20% develop venous ulcers. Therapy is required for 50% of these ulcers for more than 1 year with an estimated annual treatment expenditure of $3 billion [19,20]. To appreciate the serious sequelae of CVI it is worthwhile to examine normal venous physiology. The venous system of the lower extremities is divided into superficial and deep systems with an extensive series of perforator veins connecting the two (Fig. 2). All veins have delicate bicuspid valves that, when competent, allow venous blood to flow in one direction, returning deoxygenated blood to the central circulation.

CVI generally results from venous valve dysfunction causing pump failure, venous hypertension, and reflux [21–23]. Valve dysfunction usually originates in the deep system. Deep venous thrombosis (DVT) is a major cause of valve destruction. Initially the DVT causes obstruction to flow but in the process of recanalization valves are damaged, yielding essentially open tubes [24]. Five years after a significant DVT, 40% to 70% of patients have signs and/or symptoms of venous insufficiency as described in Box 2.

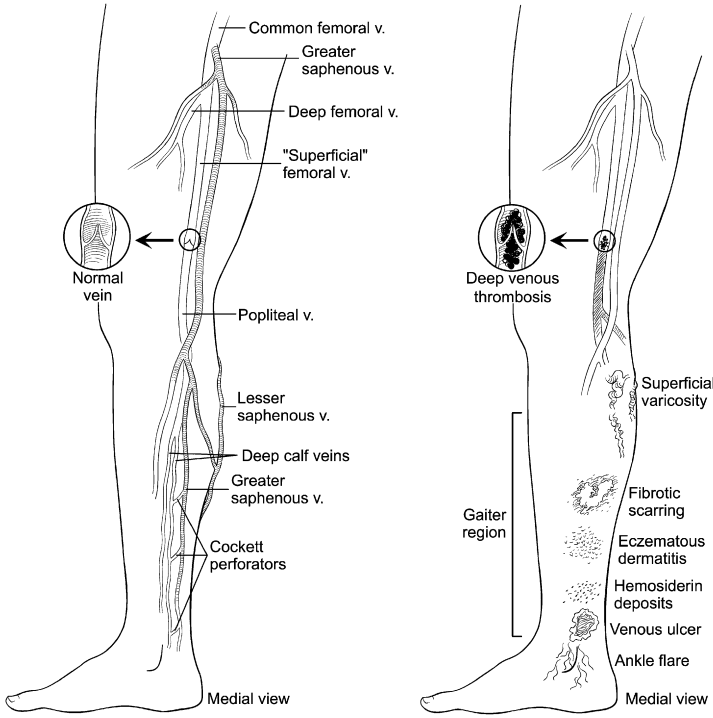


Fig. 2. The deep venous system is well supported within the muscle fascia. Disrupted deep valves allow pressure transmission through perforators to vulnerable superficial veins with resultant skin changes.

### *Pressure ulcers*

A pressure ulcer, also termed decubitus (Latin for ''lying down'') ulcer, is a potentially avoidable lesion that is frequently seen and unfortunately occasionally develops in the emergency department setting. An estimated 2.5 million pressure ulcers are treated in acute care settings per year, costing billions of dollars [25]. An individual pressure ulcer may cost up to $60,000 to treat [26]. Some 60% of pressure ulcers develop during acute care hospitalizations [27], with patients more than 70 years of age and those who have spinal cord injuries accounting for the majority. The foundation of a pressure ulcer may develop following 2 hours of unmitigated soft tissue compression [28] but may not be clinically evident for 2 to 7 days. Risk factors for developing decubitus ulcer are listed in Box 3.

Pressure ulcers occur when pressure exerted on the tissues is greater than the pressure at the capillaries. This creates an area of ischemia. If the area of ischemia is prolonged an ulcer develops. Offloading the area after prolonged pressure can also result in damage from reperfusion and the release of toxic substances that arise from the initial inflammation response. The release of

**Box 2. Chronic venous insufficiency**

*Symptoms*
- Leg heaviness, aching pressure, throbbing burning pain
- Nocturnal leg cramps, restless legs
- Discomfort worse in dependent position

*Signs*
- Ankle edema
- Superficial varicosities
- Hyperpigmentation (hemosiderin deposition)
- Eczematous skin changes
- Lipodermatosclerosis (inflamed woody induration)
- Gaiter area ulcer (usually above medial malleolus)

these substances can expand the area of damage beyond the area of pressure. Muscle tissue needs more oxygen than skin and shows greater damage and tissue destruction from prolonged pressure. A small area of pressure on the skin is often concealing a deeper area of damage beneath.

The early pressure ulcer may be difficult to detect clinically. The underlying oxygen-hungry muscle and dermis are more susceptible to ischemic damage than the relatively resistant epidermis. A stage 1 pressure ulcer may present with nonblanchable erythema and intact skin similar to a bruise. This pre-ulcerative state is vital to recognize before further breakdown occurs. The stages of pressure ulceration are outlined in Box 4.

**Emergency department evaluation**

Although chronic wounds are by their very nature slowly progressive, complications can be emergently life and limb threatening. The most serious

**Box 3. Pressure ulcer risk factors**

- Immobility (inability to effectively shift weight)
- Paralysis (spinal cord, postintubation)
- Incontinence (excessive moisture)
- Altered mental status
- Exposure to hard surfaces, friction, shear forces
- Poor nutritional status/low protein diet
- Existing pressure ulcer
- Dry sacral skin
- Nonblanchable erythema of intact skin (Stage 1 ulcer)

**Box 4. Pressure ulcer staging**

Stage 1. Skin intact but reddened (nonblanchable erythema) for greater than 1 hour after pressure is relieved
Stage 2. Blister or abrasion up to dermal layer
Stage 3. Extension of ulcer into muscle layer
Stage 4. Extension into bone or joint

threats that the emergency physician must evaluate for include overwhelming sepsis, deep venous thrombosis, pulmonary embolus, advancing local tissue destruction, and stress-induced exacerbation of existing medical conditions.

Once the ABCs of the primary survey are complete a more thorough history and physical examination may proceed. The history should focus initially on the chief complaint, which may or may not explicitly be the chronic wound itself. The practitioner must sort out what role the ulcer is playing in the patient's presenting concern. Occasionally a patient may be unaware of the ulcer's existence.

*History*

The history is then focused on the ulcer and its consequences. A detailed review may suggest ulcer cause as outlined in Table 1. Is there a past history of ulcers? How were they managed? What is the time course of the current ulcer? What therapy has already been tried and has it been effective? Is the

Table 1
History pearls in chronic wound epidemiology

| | Location | Appearance | Pain? | Findings | Therapy |
|---|---|---|---|---|---|
| Venous | Medial leg above malleolus | Large but shallow, irregular border | Some, relieved with elevation and walking | Characteristic skin changes, edema | Graduated compression, elevation |
| Diabetic | Plantar surface foot, metatarsals | Punched out | None | Absent ankle reflexes, insensate | Total contact casting |
| Pressure | Sacrum, heel, occiput | Initially a "bruise" | Yes | Immobilized elderly, spinal cord injury | Offloading |
| Arterial | Distal foot, toes | Irregular edges, little bleeding with débridement | Severe, especially with elevation and walking | Hairlessness, absent pulses, claudication, impotence | Revascularize |

ulcer changing? Is it growing, developing redness, lymphangitis, exudate, or a foul order? Is the ulcer bed friable? Is it painful? Are there associated fevers or chills? Is there a personal or family history of superficial or deep vein thrombosis? Does the patient have a known clotting disorder? Is there associated edema, leg heaviness, leg aching with prolonged standing, restless legs, night cramps, skin changes (hyperpigmentation, eczema, scarring), pruritus, or burning, all suggestive of CVI [29]? Is the discomfort relieved by elevation and walking, as in CVI, or worsened, as in arterial insufficiency (although arterial and venous disease may coexist)? Is there claudication, impotence, or generalized atherosclerotic cardiovascular disease suggesting arterial insufficiency?

*Physical examination*

The physical examination must be thorough and not be limited to the ulcer. The patient is generally assessed with focus on overall robustness, mobility, nutritional status, and hygiene. Patients who develop chronic ulcers are generally in poor health and need a comprehensive examination. Special attention is given to the ulcer, documenting the precise location, size, and depth. The gaiter area, in particular above the medial malleolus, is typical for venous ulceration. An isolated lateral malleolus rarely results from venous disease alone and may suggest a combination of trauma (car door versus driver's ankle) and arterial insufficiency. Is the ulcer mobile or is it fixed to deeper layers of tissue? The ulcer base is evaluated for purulent discharge and necrotic tissue and may be probed to determine if bone is contacted. One study indicated palpable bone protruding from the ulcer base was highly correlated with osteomyelitis [30]. The skin around the ulcer is assessed next. Is there evidence of surrounding inflammation, dilated venules, edema, infection, eczema (stasis dermatitis), hemosiderin deposits, lipodermatosclerosis, or atrophie blanche (white scarring at site of previous ulcer) seen in CVI? Is there callus developing at pressure points?

The examination continues with a more extensive vascular examination. Are pulses palpable? It is also important to calculate ankle brachial index (ABI), which is the ratio of lower extremity to upper extremity systolic blood pressure. Are varicose veins present? (Normally only veins in the foot and ankle are visible.) Is there evidence of superficial thrombophlebitis? This condition, particularly when it appears in the proximal greater saphenous vein, may lead to or concur with DVT in up to 40% of cases [31]. Has the patient experienced a sudden swelling in the leg suggestive of DVT? Even pregnant women (who typically develop venous varicosities) should be considered for DVT when sudden, unilateral swelling crops up.

Finally the neurologic examination is performed. The patient is carefully screened for neuropathy. Is the skin unusually dry? Is there muscle wasting? Are the motor strength, mobility, and flexibility within an expected range? Is the gait normal? Do the shoe soles reveal an abnormal wear pattern? Are

reflexes normal? Is the patient able to identify movements of the toes? Is the gross sensation normal? The Semmes-Weinstein monofilament is useful for assessing subtle sensory deficits. The filament is gently pressed against the distal foot until it buckles. At this point a standardized pressure has been applied. A normal person can detect a 4.17 monofilament, which exerts 1 g of linear force. The patient who is unable to sense the 5.07 mm strand, which exerts 10 g of linear force, is at markedly increased risk for neuropathic ulceration [32].

### *Diagnostic testing*

#### *Blood work*

No laboratory tests provide a direct indication of the status of a nonhealing ulcer. An elevated white blood cell count with increased band forms may indicate infection or inflammation. An abnormal erythrocyte sedimentation rate or C-reactive protein may suggest a deeper process, including severe cellulitis or osteomyelitis. The total protein and prealbumin (half-life 2 days) level give an indication of the nutritional status of the patient, a vital and often overlooked factor in wound healing. The d-dimer value is a nonspecific finding; when in the normal range it may suggest the absence of DVT or pulmonary embolus in a low- to moderate-risk patient [33]. A plasma zinc level may be indicated in nonhealing wounds [34]. Blood cultures are useful if sepsis is suspected.

#### *Microbiology*

All ulcers are colonized by bacteria. Colonization is described as a complex polymicrobial landscape that lives in balance with host defenses, generally without ill effect. The presence of excessive moisture, low oxygen tension, necrotic tissue, and dead space may tip this balance toward a more aggressive critical colonization leading to infection [35]. Infection causes persistent inflammation and leads to formation of microthrombi, causing further ischemia, increased friability, and necrosis [36].

Culture results of superficial swabbing from the ulcer base may be misleading. Other techniques, such as deep tissue sampling of an infected ulcer obtained by curettage of the ulcer base after wound débridement, deep needle aspiration, or 2-mm punch biopsy, are considered more valuable, although they may be more uncomfortable for the patient [37].

#### *Imaging*

Plain radiographs are a reasonable screening tool for a patient who has a nonhealing wound. Unsuspected foreign bodies, fractures, and gas from gangrenous infection may be visualized. The plain film may also detect osteomyelitis with its characteristic soft tissue swelling, focal demineralization, periosteal elevation, and cortical disruption. Plain radiography is not a very sensitive (60%) or specific (65%) for osteomyelitis, however. If osteomyelitis

is suspected, MRI, especially when enhanced with gadolinium, is more than 95% sensitive for soft tissue and bone inflammation [38].

Ultrasound is a widely available instrument, valuable in the evaluation of the nonhealing wound. Doppler flow ultrasound has a sensitivity/specificity for identifying arterial occlusive lesions of approximately 80% to 90% [34]. Wounds of all types depend completely on the availability of fresh oxygenated blood for healing. Doppler studies are noninvasive and may be followed by either magnetic resonance angiography (MRA) or contrast arteriography if angioplasty, stenting, or bypass are considered. Contrast angiography, although still considered the gold standard [10] of pre-interventional imaging, has significant disadvantages compared with minimally invasive MRA. These drawbacks include local arterial injury, pseudoaneurysm, traumatic AV fistula formation, thrombosis, contrast allergy, and contrast nephrotoxicity.

## Management of chronic nonhealing wounds

Initially emergency medical providers must address and manage the underlying causes of ulceration, in particular vascular insufficiency, continued trauma, and hyperglycemia. If the wound is progressively worsening despite aggressive care or is located in an atypical location, other possibilities should be considered. These diagnoses include underlying malignancy [39], vasculitis, atypical infection, shooter's patch (an area exploited for repeated vascular access by intravenous drug user) [40], foreign body reaction, nonaccidental trauma [41], and factitious wound. The general principles of chronic wound treatment are cleanse, nourish, and protect.

### *Cleansing*

All wounds should be cleansed as thoroughly as possible without disturbing or disrupting underlying viable tissue. Initial decontamination is performed using high-pressure irrigation as for any wound (saline lavage using a large syringe with 18-gauge needle or anti-splash attachment). Topical disinfectants, such as hydrogen peroxide, iodophors, and chlorhexidine, are not generally used in management of chronic ulcers. They may hamper wound healing by direct cytotoxic effects on keratinocytes and fibroblasts. Gentle patting of the surface with soft moist gauze may follow. Further cleaning occurs with débridement (ie, the removal of nonviable tissue and, in the latest understanding, disruption of the biofilm, a protective polysaccharide layer secreted by bacteria). In general, a lower rate of healing is achieved in wounds that are débrided less frequently [42]. In the emergency department sharp débridement is performed with steel scalpel, scissors, and forceps. Sharp débridement is aimed at removing overtly necrotic material and fibrinous exudate. If a stable eschar (ie, a hardened crust of dead tissue) has formed it may not need to be removed. Sharp débridement is selective

and controllable. Care must be taken to avoid healthy tissue, and liberal use of local or regional nerve anesthesia is useful for extensive débridement.

*Nourishment*

Nourishment is the second feature of therapy. Wounds are nourished by blood, oxygen, moisture, food, and external factors. Most important by far is ensuring an adequate arterial blood supply to the wound. The ABI has been shown in multiple studies to be a highly predictive tool [43]. ABI has a roughly 95% sensitivity and specificity for angiographic peripheral arterial disease when less than 0.9 [44]. A value greater than 1.2 may be falsely elevated, a result of calcified lower extremity vessels. A value of between 0.7 and 0.9 indicates a mild to moderate degree of peripheral arterial disease. Anything less than 0.7 denotes significant pathology and should be referred to a specialized vascular laboratory for more sophisticated evaluation, including transcutaneous oxygen tension (TCOT) and possible angiography. ABI reflects macrovascular arterial status, whereas TCOT reveals macro- and microvascular conditions. A patient who does not have diabetes requires a minimum TCOT of 30 mm Hg to heal a wound [12]. Patients who have diabetes require a level of 40 mm Hg to heal.

Adjunctive hyperbaric oxygen therapy (HBOt) has been shown to be effective in healing wounds that are reversibly hypoxic as demonstrated by TCOT. This process can elevate local tissue oxygen tensions, resulting in improved leukocyte function, enhanced collagen synthesis, and neovascularization. It has also been shown to inhibit anaerobic bacterial activity.

Another vital nourishing factor is moisture. Studies since the 1960s have repeatedly indicated a moist environment is significantly better than dry milieu for healing [45–48]. Conversely, dehydration by air exposure, dry gauze, heat lamps, and so forth, is detrimental [28]. Topical moisturizing preparations vary widely. In general, simple petrolatum without preservatives is best. Lanolin may over time become a sensitizing agent. Other over-the-counter products may be helpful but many contain sensitizing agents and, in an individual patient, should be discontinued if skin irritation arises.

Several other nourishing factors have been studied. Topical therapies, including recombinant growth factors (which stimulate angiogenesis, collagen formation, and epithelialization), bilayer keratinocyte skin grafts, and cadaveric skin transplants, are available [49]. Vacuum assist devices clearly aid formation of granulation tissue in difficult wounds [50]. Vitamin deficiencies must be considered and addressed. A daily diet rich in protein, approximately 1.5 to 2 g/kg body weight, is critical for wound healing [51].

*Protection*

Protection is the third pillar of wound healing. The wound must be physically protected from additional trauma and injury and failure to do so

negatively affects the healing process. Pain is an indication of underlying pathology that may be reversible. Bacteria release mediators of inflammation that sensitize pain receptors. Bacterial bioburden may be the most common reason for pain within a wound. Simple, sharp débridement often improves this discomfort. Unrelieved pain may require topical or systemic medication and may necessitate specialty pain management referral. Unmitigated pain leads to decreased patient compliance, depression, and overall worsened quality of life.

### *Specific recommendations*

#### *Chronic venous ulcers*

The foundation of CVI ulcer therapy is compression and elevation [52,53]. A wound in the normal healing process develops swelling, a localized edema fluid. If this expected edema stagnates and accumulates, as in the case of CVI, it becomes saturated with proinflammatory factors that lead to impaired healing and secondary lymphedema, occasionally massive. Initially when a patient presents to the emergency department with uncontrolled edema, the practitioner focuses on limb volume decompression. Tubular elastic bandages are a simple and cost-effective product to get the process started. The patient should be encouraged to keep the legs elevated above chest level while sleeping and to avoid dangling legs unsupported while sitting. Elevation of the affected lower extremities offers symptomatic relief and improved healing.

Graduated compression stockings are a prescribed, measured, and expensive product used to keep edema at bay. Graduated compressive leg garments are not to be confused with the commonly available, all-purpose stockings, which offer little benefit. Patients who have arterial insufficiency, especially if the ABI is less than 0.5–0.8, may not be good candidates for compression. Stockings are divided into areas of progressively diminishing compression starting with highest pressure at the ankles (20 to 30 mm Hg in moderate disease and 30 to 40 mm Hg in more severe disease) and lowest pressure at the upper end. The stockings may be knee high or extend to the thigh. These compressive dressings can be used daily for 6 to 9 months before losing effectiveness. A word of caution: patients occasionally do not tolerate the thigh-high stockings and may roll them down for comfort. This may create a tourniquet effect and be unsafe.

Stretchy elastic bandages have been used as compressive dressings. Caution must be advised not to place them too tightly or without gradually diminishing pressure as they run upward to avoid a tourniquet effect. They are considered by some to be too unpredictable in their action. In addition, they tend to lose their elastic quality quickly.

An Unna boot is a gauze wrap impregnated with zinc oxide and calamine. As it dries, it becomes a rigid support and thus acts as a firm strut against which the calf muscles contract. This product has been available

for many decades and its mechanism of action is not clearly understood. There are many varieties of multilayer compression bandage systems that provide excellent support and compression. The surrounding skin of a venous ulcer frequently exhibits signs of eczematous dermatitis that may respond to a topical steroid preparation.

*Diabetic ulcers*

Treatment of diabetic foot ulcers should be aimed at treatment of neuropathy, foot deformity, repetitive trauma, poor glycemic control, and arterial insufficiency. Each of these factors should be addressed in treatment. Foot ulcers are often the precursor to amputation and thus must be treated aggressively. Appropriate referrals to the endocrinologist, podiatrist, wound care specialist, and vascular surgeon should be considered.

Pressure relief is essential in the treatment of ulcers. Total contact casting (TCC) is a specialized casting method that allows continued ambulation while offloading the ulcer [54]. TCC is changed weekly but is not removable by the patient, thus ensuring compliance. Removable TCC variations have been shown to have less effectiveness because of patient nonadherence [55]. The removable TCC can be converted to nonremovable with the simple addition of wrapped cohesive bandage and plaster of Paris. Diabetic ulcers that have been resistant to other forms of therapy may heal within 6 weeks with TCC [56]. Pressure relief in less severe circumstances may be achieved with computer-designed footwear. If custom footwear is not an option for the patient, quality athletic running shoes or shoes with a larger toe box may be an alternative. All shoes should be fitted in the later afternoon when foot swelling is greatest. Therapeutic shoes bring about a dramatic decrease in ulcer recurrence rate. Foot deformities may be referred to a podiatrist to evaluate appropriateness of prophylactic surgery.

Diabetic ulcers are at high risk for polymicrobial infection with an average of 1.5 to 5.8 bacterial isolates per patient, two thirds aerobic and one third anaerobic [37]. *Staphylococcus aureus* is the most common isolate, with an increasing percentage methicillin resistant [57,58]. Group B β-hemolytic streptococcus is another common and potent pathogen. Gram-negative organisms are also pervasive. Ulcer therapy usually requires a combination of agents. Severe infection may respond to combination vancomycin plus β-lactam/β-lactamase inhibitor or vancomycin plus carbapenem [59].

If the condition is clinically appropriate for outpatient management, the patient should be instructed to monitor the condition closely and should be given appropriate referral for reevaluation and continued wound care. If that is not considered feasible, the patient should be instructed to return to the emergency department in 24 to 48 hours for reevaluation. These wounds may worsen quickly leading to osteomyelitis and bacteremia. Osteomyelitis complicates diabetic ulcers in one third to one half of severe ulcers and requires prolonged and specialized care.

*Pressure ulcers*

Pressure ulcer therapy begins with offloading of the lesion. For patients who are completely immobilized, particularly on a standard surface, turning and repositioning should occur on a frequent basis, at least once every 2 hours. Sophisticated and expensive dynamic air-fluidized support systems are available and may be superior to the less expensive but more readily available static support mattress overlays. There are multiple pressure-reducing devices that can be compared in effectiveness [25,60].

Aside from essential reduction of skin friction and shear forces, the wounds should be kept clean, well débrided, moisturized, possibly dressed with occlusive dressings, and free of active infection. Although moisture is an important element in wound healing, excessive moisture in the form of sweat, exudate, or incontinence is linked to wound aggravation. Topical or oral antibiotics are useful in low-grade infection, especially if *Staphylococcus*, *Pseudomonas*, *Providencia* or anaerobic species are cultured [28].

*Dressings*

There are many basic types of dressings, some with embedded antimicrobials, categorized into hydrogels, alginates, hydrocolloids, foams, films, and others, each with advantages and disadvantages [53,61,62]. A particular dressing is selected by a wound care specialist based on the specific features of the existing condition. If the wound is essentially dry and clean, nonadherent, soothing, water-soluble, carboxy-methylcellulose hydrogel can be applied directly to the wound [63]. This is wrapped with dry gauze. If the wound is seeping abundant exudate, an absorptive calcium alginate dressing may be more appropriate. Hydrocolloids protectively adhere to a shallow wound, promote autolysis and granulation tissue, and are impermeable to moisture, bacteria, and contamination. Absorbent, opaque, nondébriding foams and transparent nonabsorbent films also are available.

*Antibiotic coverage*

Antibiotic coverage should be directed primarily toward *S. aureus* (including methicillin resistant) and gram-positive *Streptococcus* (especially β-hemolytic). Many wounds have polymicrobial pathology involving anaerobes, *Enterobacter*, *Pseudomonas*, and a host of less common pathogens [58,64–66]. Appropriate antibiotic combinations found in major antimicrobial guides are then fine honed based on local community sensitivity patterns. Agents, often used in combination, include cephalosporins, β-lactam/β-lactamase inhibitor, fluoroquinolones, clindamycin, imipenem/cilastin, vancomycin, metronidazole, linezolid, and daptomycin. Duration of therapy is 7 to 10 days in mild to moderate infection and as long as necessary in more critical circumstances. Topical antibiotics used alone or in combination may be effective in low-grade infection. Silver sulfadiazine 1% cream (broad antimicrobial), mupirocin (only effective against

gram-positive cocci), polymyxin (gram-negatives, including *Pseudomonas*) are generally well tolerated. Topical neomycin (effective against many gram-negative species and staphylococci but not *Pseudomonas*, *Streptococcus*, or anaerobes) and bacitracin (gram-positive activity) may be allergenic in approximately 10% of patients [67]. Patients who have arterial insufficiency or moderate to severe infection require parenteral preparations and vascular surgery consultation.

## Summary

Chronic wounds are an immense source of suffering, health care expenditure, and disability for literally millions of patients. On essentially a daily basis, a patient who has either a chronic nonhealing wound or is at risk for one presents to every Emergency Department (ED) in the country. The ED practitioner is in a unique position to identify these patients, initiate appropriate care, and establish proper referrals. Specialists within the field of chronic wound management are making enormous advances implementing sophisticated care but often are unable to make contact with the population at risk. With sensitivity to these conditions, the vigilant ED practitioner can have an enormous impact on public health.

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